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THE MANAGEMENT OF HEART BLOCK

BY

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In 1952, Zoll described a technique for artificial pacing of the heart. The method employed electrodes placed on the chest wall and electrical impulses of 2 msec. duration at 25–150 volts. Although this indirect technique proved successful in resuscitating and temporarily maintaining life in patients with Stokes-Adams attacks due to asystole, the high voltage required produced skin burns and painful contractions of the muscles of the thorax. Other methods were developed for the direct stimulation of the myocardium with low voltages, and permanent pacing of the heart with small pacemakers is now routinely performed (Bellet *et al.*, 1960; Chardack, Gage, and Greatbatch, 1961; Elmqvist and Senning, 1960; Davies, 1962; Kahn *et al.*, 1960; Landegren, 1962; Lillehei *et al.*, 1960; Stephenson *et al.*, 1959; Weirich *et al.*, 1958; Zoll *et al.*, 1961). Reports on the success of long-term pacing by many different systems have now appeared (Furman *et al.*, 1961; Levitsky *et al.*, 1962; Siddons, 1963). While artificial pacemaking is life saving and permits a return to a normal way of life in most cases, numerous problems have arisen in the maintenance of this artificial system. It is for this reason that we propose to describe our experiences in the management of heart block over the past four years at St. George's Hospital, of which some have been previously reported by Portal *et al.* (1962) and Siddons (1963).

PATIENTS

Of the first 100 patients who were paced, after medical treatment had failed, 51 were males aged between 4 and 82 years (mean 62·5 years), and 49 were females aged between 27 and 84 years (mean 64·2 years). In addition, there were 40 patients (28 males between 21 and 85 years (mean 69·7 years) and 12 females aged 12 to 85 years (mean 64 years)), who were thought not to need artificial pacing, since their symptoms were mild and they were maintained on medical treatment during the same period.

By April 1964 a total of 88 patients had been treated by long-term artificial pacing and the remaining 12 by temporary pacing only. The length of time the long-term patients have been paced ranged from 1 day to 48 months (mean 12·7 months). The temporary group were paced for 1 to 14 days (mean 5·5 days). Twenty-one patients on long-term pacing have died (28%) as have 7 from the temporary group (58%). Of the 40 patients managed on medical treatment alone over a four-year period, 12 have died (30%). The indications for pacing are shown in Table I.

AETIOLOGY

Apart from 5 patients who were paced temporarily following acute myocardial infarction and 1 patient who developed complete heart block following surgical repair of Fallot's tetralogy, the cause of the heart block was usually obscure. Of the remaining 94 patients with chronic block who were

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TABLE I

INDICATIONS FOR ARTIFICIAL CARDIAC PACEMAKING

	No. of patients
Stokes-Adams attacks uncontrolled by medical treatment	59
Stokes-Adams attacks with congestive cardiac failure uncontrolled by medical treatment	24
Stokes-Adams attacks with congestive cardiac failure and mental confusion uncontrolled by medical treatment	5
Congestive cardiac failure secondary to slow rate uncontrolled by medical treatment	7
Congestive cardiac failure and mental confusion uncontrolled medical treatment	2
Heart block following correction of Fallot's tetralogy	1
To cover unrelated surgical procedures after previous incident of cardiac arrest	2

TABLE II

"RELEVANT" PAST MEDICAL HISTORY IN 94 PACED PATIENTS WITH CHRONIC BLOCK

	No. of patients
Diphtheria	11*
Rheumatic fever	4
Rheumatoid arthritis	4
Hypertension	3†
Diabetes	3
Paget's disease	1
Alcoholism	1
Myxœdema	1

* 32 patients possibly not asked.
† Diastolic pressure above 100 mm. Hg before pacing.

paced, the incidence of "relevant" illness in their past medical history is shown in Table II. The incidence of diphtheria (17·7%) appears to be high, but its true significance is difficult to assess in the absence of a suitable control population. Diphtheria was a fairly common disease during the childhood of these patients and so a moderate incidence in the series would be expected; it was certainly not significant ætiologically in the majority of cases, though it has been suggested as a cause of heart block developing in later life (Butler and Levine, 1929; Penton, Miller, and Levine, 1956). Rheumatic fever is unlikely to play a part in the development of heart block except where valvular disease has been produced in later life (Penton *et al.*, 1956; Rowe and White, 1958). In our series, 3 patients had aortic stenosis and 1 had mitral stenosis. Rheumatoid arthritis (4·2%) is not infrequently associated with myocardial lesions and may play a part in the development of heart block (Brooks *et al.*, 1955; Olhagen, 1960). The incidence of other diseases listed in Table II is probably no greater than in the general population.

The part played by coronary artery disease in the development of chronic heart block has been overemphasized. In our series of 100 long- and short-term paced patients, 28 have died; 19 had post-mortem examinations carried out with injection of the coronary vessels and some histological examination of multiple sections of the myocardium: of these, 15 had normal coronary vessels and the remainder had a varying degree of severity of coronary artery disease. Table III shows the state of the coronary arteries at necropsy and the electrocardiogram in these 19 patients. There was a high incidence of right bundle-branch block pattern QRS complexes both with conducted and idioventricular beats, including the 4 patients with coronary disease. In addition, 27 patients gave a history suggestive of ischæmic heart disease, yet analysis of their records revealed no evidence of ischæmic changes apart from 6 patients, who had left bundle-branch block, making interpretation difficult.

The present findings are in keeping with reports of isolated fibrosis of the conducting system unrelated to coronary artery disease (Penton *et al.*, 1956; Shirley Smith and Zoob, 1961; Johansson, 1961; Portal *et al.*, 1962; Dittmar, Friese, and Holder, 1962).

TABLE III

STATE OF CORONARY ARTERIES *POST MORTEM* IN 19 PATIENTS AND THEIR ELECTROCARDIOGRAMS

State of coronary arteries	No. of patients	E.C.G. pattern
Severe coronary artery disease	1	Complete block with slow nodal rhythm
Slight coronary artery disease	2	Complete block with right bundle-branch block idio-ventricular rhythm
Anterior cardiac infarction, coronary embolus and calcified mitral annulus	1	Sinus rhythm with right bundle-branch block
Normal coronary vessels	15	Complete block with right bundle-branch block idio-ventricular rhythm in 6; complete block with slow nodal rhythm in 4; sinus rhythm with right bundle-branch block in 5

CLINICAL AND ELECTROCARDIOGRAPHIC FEATURES OF 100 PACED PATIENTS

Attacks of unconsciousness occurred in all but 12 patients and 5 had been treated for epilepsy before the correct diagnosis was made. Cardiac failure had occurred in 38 patients and was the only indication for artificial pacing in 7 of them (Table I).

The electrocardiograms are analysed in Table IV. The preponderance of patients with right bundle-branch block was striking and may be related to the anatomical arrangement of the fibres of the conduction tissue. A lesion near the atrio-ventricular node might interrupt conduction in the whole of the right bundle where the fibres are closely packed, but it might initially spare much of the left bundle which spreads out over a wide area. This would apply whether the stimulus arose in the sino-atrial or atrio-ventricular nodes. In all, 71 of the 100 patients (71%) had a QRS complex of right bundle-branch block pattern, compared with an incidence of 27 per cent reported by Penton *et al.* (1956), but patients who manifested only first or second degree atrio-ventricular block with or without syncope were not included in their study.

In the majority of the patients who were having Stokes-Adams attacks, the underlying mechanism was asystole, but some patients lost consciousness from episodes of ventricular tachycardia or short episodes of ventricular fibrillation, though this was nearly always preceded by a slow heart rate (Table V). It is probable that the true incidence of fast heart rates as a cause of Stokes-Adams attacks is higher than we were able to record with only short periods of monitoring. A history of palpitations in patients with Stokes-Adams attacks should always be regarded as suspicious of episodes of ventricular tachycardia, and the institution of sympathomimetic drug treatment outside hospital as unwise. One patient in our series, during a period of continuous monitoring, developed

TABLE IV

ANALYSIS OF ELECTROCARDIOGRAM IN 100 PATIENTS PRIOR TO PACING

	No. of patients
Complete heart block, idioventricular rhythm	81
Right bundle-branch block pattern	58
Left bundle-branch block	12
Varying right and left bundle-branch block	2
Sinus rhythm periodically becoming complete heart block	19
2:1 block with normal Q.R.S.	2
Prolonged P–R interval with right bundle-branch block	1
2:1 block and 6:1 block with right bundle-branch block	2
Normal P–R interval with right bundle-branch block	9
Normal P–R interval with left bundle-branch block	4
Normal P–R interval with varying left and right bundle-branch block	1

TABLE V

RHYTHM CAUSING STOKES-ADAMS ATTACK IN 100 PATIENTS

	No. of patients
Asystole alone	67
Asystole with ventricular tachyarrhythmia	16
Ventricular tachyarrhythmia alone	7

ventricular tachycardia and lost consciousness shortly after beginning long-acting isoprenaline therapy. In the series of Parkinson, Papp, and Evans (1941), 45 per cent of attacks were due to ventricular tachycardia with or without subsequent standstill. Similar results have been reported by Johansson (1961) and DeSanctis (1963).

METHODS OF ARTIFICIAL PACING

External Pacing. In extreme emergency, stimuli of up to 200 volts were applied across the intact chest, using a simple condenser discharge unit (Leatham, Cook, and Davies, 1956), and when used promptly on patients with asystole, never failed to start the heart. But this method is of course extremely painful on the return of consciousness. Should the external pacemaker be required for any length of time to maintain an adequate heart rate, an electrode catheter must be passed from a peripheral vein and wedged at the apex of the right ventricle. At present we use a No. 5 solid unipolar catheter (C.50), made by the United States Catheter Corporation. The current bipolar electrode catheters have not proved satisfactory, due to fractures of the electrode wires.

Internal Pacing. With the exception of 3 units of the type recommended by Elmqvist and Senning (1960) all our units were designed by one of us (G.D.). During development, the design of the units has been altered and their relative merits have been described (Siddons, 1963; Davies and Sowton, 1964). The majority of units implanted have been of the constant voltage type, powered by four mercury batteries potted in an epoxy resin; some were coated with silicone rubber. They were sterilized by chlorhexidine fluid and more recently by formalin vapour. These pacemakers give a square wave impulse of 1–2 msec. at a fixed rate, the no-load voltage being 5 volts and the estimated battery life 3 to 5 years. The pacemakers are normally adjusted to deliver 65–75 impulses per minute. There is good evidence that at this rate the stroke volume of the heart can be increased to cope with the demands made by exercise (Chardack, 1964 a, b; Sowton, 1964; Judge, Wilson, and Siegel, 1964).

The method of implanting the abdominal pacemaker with the location of the electrodes on the myocardium has been described by Siddons (1963) and was the initial method of long-term pacing in 59 patients (Table VI). Eighteen patients have been maintained on endocardial electrode pacing inserted via the jugular venous system, with the electrode catheter exteriorized above the clavicle and the positive electrode buried in

TABLE VI

PRESENT STATUS OF 59 PATIENTS TREATED WITH ABDOMINAL-EPICARDIAL SYSTEM

	No. of patients
Implanted pacemaker functioning satisfactorily	15
Functioning but exteriorized pacemaker	9
No longer paced	1
Transferred to endocardial-jugular system with implanted pacemaker	9
Transferred to endocardial-jugular system with external pacemaker	8
Deaths	17

the subcutaneous tissue of the neck; the pacemaker was attached to the electrode catheter and positive electrode externally. There were 28 patients who were treated with an endocardial electrode inserted via the jugular venous system and the electrode and attached pacemaker were buried in the axilla (Siddons and Davies, 1963). Some have been paced for periods of time by more than one of these methods (Table VI).

RESULTS

When artificial pacing had been satisfactorily established, all episodes of loss of consciousness were abolished. In many instances congestive cardiac failure was relieved and intellectual function improved. Exercise tolerance was improved, and most patients are now leading relatively normal lives. An ejection systolic murmur was heard at the base before pacing in 80 patients and was attributed to increased stroke volume in all but 3 who had aortic stenosis in addition to heart block. After pacing, this murmur disappeared completely in 50 patients and was reduced in intensity in 30. The cardiothoracic ratio was greater than 1:2 in 76 patients before pacing and after pacing for a mean of 12·7 months there was no statistical difference in this ratio; but occasionally patients showed considerable improvement (Fig. 1).

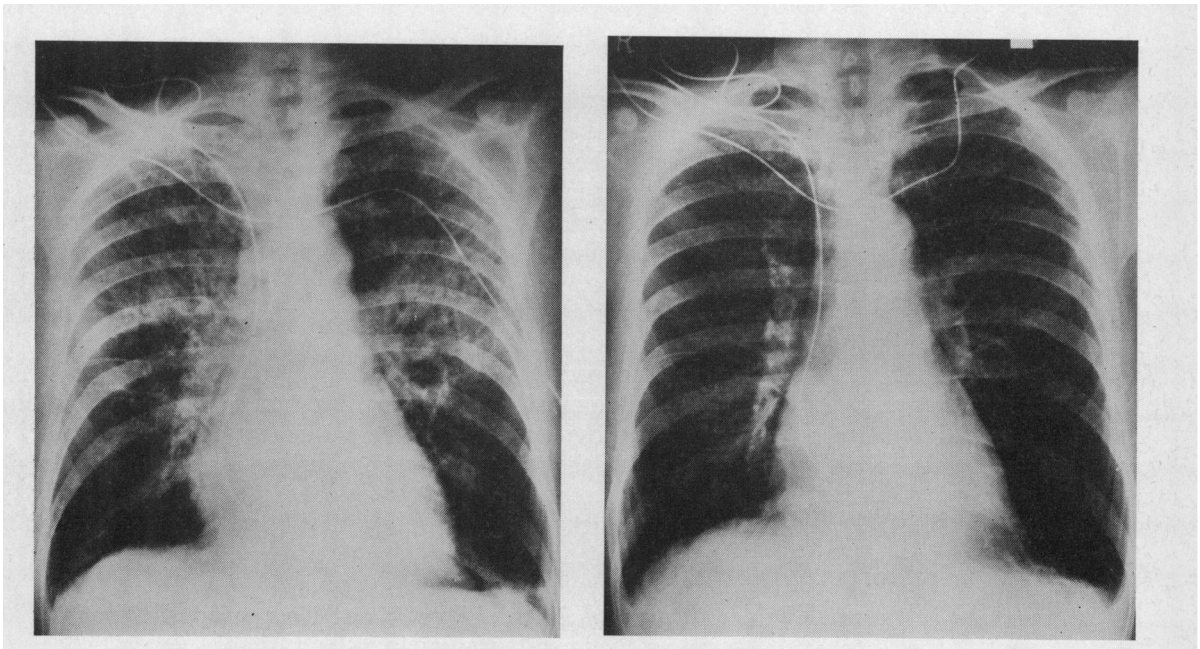

FIG. 1.—Chest radiographs before and after pacing, showing regression of pulmonary œdema and a decrease in heart size.

Statistical analysis of the alterations in systemic blood pressure produced by pacing show a mean systolic fall of 50 mm. Hg, with a mean diastolic rise of 5 mm. Hg.

There were 43 patients with blood ureas above 40 mg./100 ml., and in all of them pacing led to a fall in the level, which became normal in all but 5 patients. Similar improvement in renal function during pacing has been noted by Müller and Bellet (1961).

COMPLICATIONS OF LONG-TERM INTERNAL PACING

(1) *Epicardial System with Buried Abdominal Pacemaker.* It can be seen from Table VI that 26 (42·6%) patients had to be changed to another system; this includes 3 patients who were referred from elsewhere and 5 patients whose units were of an early design. The principal reason for the

TABLE VII

COMPLICATIONS ASSOCIATED WITH INSERTION AND REMOVAL OF ABDOMINAL-EPICARDIAL SYSTEMS IN 59 PATIENTS

	No. of patients
Pleural effusions following thoracotomy*	22
Basal collapse	16
Sepsis and/or rejection of units	24†
Serious hæmorrhage from right ventricle at thoracotomy for removal of myocardial electrodes in the presence of sepsis	2
Congestive cardiac failure	4
Pulmonary embolism	3
Renal failure	3
Pneumonia	4
Empyema	1
No complications	8

* 18 effusions persisted 4 or more weeks radiologically. The pleura was not drained at operation.
† See text.

change was the rejection of the units by the body after varying lengths of time ranging from 3 weeks to 10 months. This may have been due to a low grade infection introduced at the time of implantation, or to reaction by the tissues to a foreign body. The first sign was the development of pain at the site of the unit, and, later, sinuses appeared in the abdominal or thoracotomy scars with relief of pain. Whether infection preceded sinus formation or not, once it was established tissue changes occurred around the electrodes resulting in an increase in the power required to pace the heart (threshold). Exteriorization of the unit at this stage became necessary and enabled confirmation of the increase in threshold, which often fell for a time when drainage was established. Though many patients can be maintained for a time in this state, the threshold continues to rise, almost inevitably necessitating a change to endocardial pacing.

The complications of insertion of the abdominal epicardial system were high (Table VII). Though the majority of the complications are those associated with any thoracotomy in a group of patients largely in their sixth and seventh decade, they are complications serious enough to consider alternative methods of pacing. Once sepsis is present, the whole system of wires and electrodes may have to be removed. In 2 patients serious hæmorrhage occurred at the time of operative removal of electrodes from the wall of the right ventricle which was involved by abscess cavities.

Of the 59 patients paced with the abdominal epicardial system, 17 have died (see Table XII).

(2) *Permanent Endocardial-Jugular Pacing with External Pacemaker.* This group of 18 patients has been largely derived from patients whose abdominal-epicardial system had failed, or the patient had refused alternative methods of treatment, though initially the method was regarded as the method of choice in patients whose clinical condition was too poor for a thoracotomy. The number of patients and complications are shown in Table VIII.

The major difficulty in maintaining satisfactory pacing was to ensure that the electrode tip was not pulled out of its "wedged" position in the right ventricle; this was particularly liable to happen following removal of the plaster dressings used to hold the catheter in place above the clavicle. The incidence of the need to re-position the catheter was reduced when a loop was made in the catheter beneath the skin at its point of exit above the clavicle, though this may increase the chances of fracture of the catheter. Septicæmia occurred in 2 patients following repositioning of the electrode catheter and no doubt was due to blood-stream contamination from the minimal but inevitable area of infection at the point of exit of the catheter. One other patient, who had mitral valve disease, developed subacute bacterial endocarditis shortly after endocardial pacing was started. Although antibiotic treatment initially controlled the septicæmia in the 3 patients in this group, final cure was only achieved by removing the endocardial system.

TABLE VIII

COMPLICATIONS ASSOCIATED WITH PERMANENT ENDOCARDIAL-JUGULAR PACING WITH EXTERNAL PACEMAKER IN 18 PATIENTS

	No. of patients
Current number of patients	14
Transferred to abdominal-epicardial system	1
No longer paced	1
Septicæmia	3
Repositioning of catheter	16 times in 8 patients
Perforation of the myocardium by the electrode catheter	1
Thrombo-embolic episodes	0
Number of deaths	2

The myocardium was perforated by the C.50 (No. 5) electrode catheter in one patient, and this resulted in intermittent pacing and was possibly related to movement of the catheter tip deeper into the right ventricle when dressings on the patient's neck were changed. However, the patient was none the worse and was subsequently treated with an abdominal-epicardial system of pacing. Intermittent pacing due to inadvertent insertion of the catheter tip into the coronary sinus, which is followed by a rise of threshold, is avoided by passing the electrode tip through the pulmonary valve before withdrawing and "wedging" the tip at the apex of the right ventricle.

Two patients with intractable heart failure were uninfluenced by pacing and died within 24 hours.

(3) *Permanent Endocardial-Jugular Pacing with an Implanted Pacemaker.* The technique of installation in the axilla has been described by Siddons and Davies (1963) and a chest radiograph of a patient with the system is shown in Fig. 2. Under local anæsthesia the electrode catheter is inserted into the right ventricle via the external or internal jugular vein and the threshold for pacing is measured. Since the threshold for pacing rises tenfold to approximately 5 microjoules (3 volts), the initial pacing threshold should be less than 0·5 volts (power 0·5 microjoules). The patient is then anæsthetized and an incision made in the axilla and the proximal part of the electrode catheter is drawn down to the axilla, behind the clavicle, and attached to the pacemaker and positive electrode. The unit is then completely buried in the axilla. So far 28 patients have been treated and the complications (Table IX) have been remarkably few when compared with the abdominal-epicardial system. Two patients developed intermittent pacing four months after successful pacing had been established, and a fracture of the electrode catheter was found in the loop placed in the neck. Subsequently it has been found unnecessary to loop the catheter in the neck with this system of pacing since movement of the arm does not tend to move the catheter at the site of entry into the vein. It is important to ensure that there are no loops or redundant catheter lying in the right atrium, since this has been responsible for the catheter tip becoming displaced in 1 patient, resulting in intermittent pacing. One patient developed septicæmia following insertion of the axillary-jugular system of pacing as an emergency

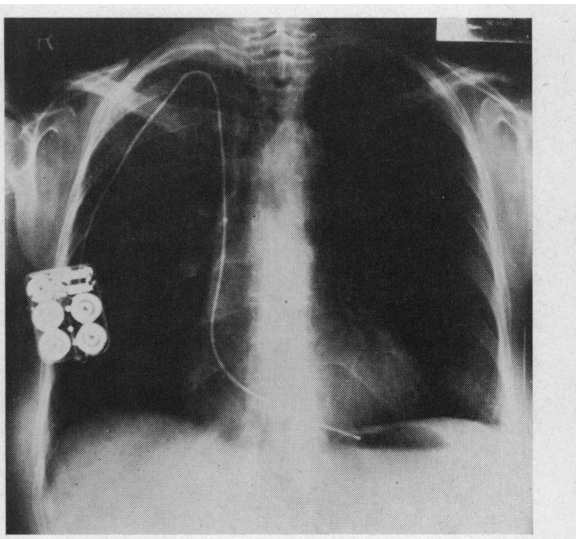

FIG. 2.—Chest radiograph showing the endocardial-jugular system with buried pacemaker.

TABLE IX

COMPLICATIONS ASSOCIATED WITH ENDOCARDIAL-JUGULAR SYSTEM WITH IMPLANTED PACEMAKER IN 28 PATIENTS

	No. of patients
Fractured catheters	2
Septicæmia and axillary abscess	1
Wound breakdown and local sepsis	2
Fragmented positive electrode	1
Transferred to abdominal-epicardial system*	4
Repositioning of electrode catheter within 24 hours	4 times in 4 patients
Late repositioning	0
Deaths	1

* See text.

procedure, and removal was necessary. Unfortunately resuscitative procedures during the operation resulted in contamination of the axilla and electrode catheter with the subsequent development of an abscess. Thus the present policy in an emergency is to pace temporarily from an arm vein and install a buried jugular system under ideal conditions later.

Endocardial pacing is entirely dependent on a stable "wedged" position of the tip of the electrode catheter in the right ventricle. Four patients paced intermittently within 24 hours of insertion of the system because the tip of the catheter had become displaced into the outflow tract of the right ventricle. The catheters were easily repositioned by opening the neck incision. Once pacing has been established without "missed" beats, unless in a refractory period, for longer than 24 hours, no displacement of the catheter tip from a satisfactory position has occurred. Faulty positioning of the pacemaking unit immediately beneath the axillary incision resulted in wound breakdown and subsequent local infection in 2 patients, but healing followed re-implantation of the unit well away from the site of the incision in one patient and in the other the catheter was exteriorized and attached to an external pacemaker.

Diaphragmatic stimulation occurred in 1 patient and the discomfort became intolerable. This was successfully treated by phrenic nerve crush.

Perforation of the heart by the endocardial electrode catheter once in position has not yet occurred using the C.50 (No. 5) catheter and a unit buried in the axilla and may be due to the greater stability within the heart of the totally implanted system. However, the heart has been perforated on two occasions at the time of insertion of the electrode catheter, and this may have been related to overenthusiastic "wedging" of the catheter tip. This resulted in a rapid rise in the threshold for pacing and was treated by repositioning the catheter without any other sequelæ.

Of the original 28 patients, 4 have been transferred to abdominal-epicardial systems of pacing. These patients were the first to be treated by the endocardial-jugular implanted unit system and the pacemakers inserted were underpowered and pacing ceased within a few days. With the use of the more powerful unit this problem has not recurred.

Only 1 patient has died and this followed a head injury sustained during a fall on an icy road. After 3 to 11 months (mean 8 months), 22 patients continue to pace satisfactorily.

(4) *Temporary Endocardial Pacing with External Pacemaker.* Five patients who developed complete heart block following acute myocardial infarcts were paced for 3 to 8 days (mean 5 days) until sinus rhythm was re-established (Table X), and the electrode catheters were left in position in the right ventricle for a further 7 days lest the heart block should recur. Nevertheless, 4 of the 5 patients subsequently died suddenly after withdrawal of monitoring, while still in hospital, presumably owing to a recurrence of block, and in future we intend to pace or monitor for 3 to 4 weeks. Six patients were paced either for assessment of improvement of confusional states associated with slow heart rates, or to cover unrelated surgical procedures after previous incidents of cardiac arrest. One

TABLE X

INDICATIONS FOR TEMPORARY ENDOCARDIAL PACING WITH AN EXTERNAL PACEMAKER IN 12 PATIENTS

	No. of patients
Complete atrio-ventricular block with Stokes-Adams attacks following acute myocardial infarction	5
Angina pectoris associated with slow nodal rhythm	1
Confusional states associated with slow rate not subsequently improved by pacing	3
To cover unrelated surgical procedures after previous incident of cardiac arrest	2
Ventricular tachyarrhythmia later responding to medical treatment	1

patient, who had aortic stenosis, developed subacute bacterial endocarditis while being temporarily paced prior to aortic valvotomy. This was before it was realized that prophylactic antibiotics were required in the presence of rheumatic valve disease with endocardial pacing. One other patient in this group was paced for 7 days following episodes of angina pectoris associated with brief periods of slow nodal rhythm and loss of consciousness. While he was paced there were no further attacks and he has subsequently remained free of symptoms.

MAINTENANCE OF ARTIFICIAL PACING

The length of life of the units has been disappointing, for it was expected to be 3 to 5 years, but in practice has averaged 12·7 months owing to premature battery exhaustion, since no account had been taken of the effect of heat from the body. This has, on occasions, led to the production of dangerous tachycardias, for the units were designed to discharge at faster rates as the batteries became exhausted. The design was altered in January 1964 so that the units gradually slow when the batteries are running down, but this too can lead to dangerous arrhythmias; therefore, all patients are taught to take their pulse daily and it should not vary more than 10 beats a minute.

In order to maintain 88 long-term artificial-pacing patients, 128 units have been required for implantation in the abdominal wall or axilla. The reasons for removal of the units are shown in Table XI. The commonest causes of unit failure were faults in the transformers and transistors, which, it is hoped, have now been eliminated. The life of the batteries can be expected to be

TABLE XI

ANALYSIS OF FATE OF 128 PACEMAKERS IMPLANTED IN 88 LONG-TERM PACED PATIENTS

	No. of standard units	No. of prototype units
Pacing satisfactorily	37	0
Sepsis and/or tissue reaction	20	4
Battery exhaustion*	11	1
Patients dying with satisfactory unit *in situ*	11	4
Broken electrodes (includes 2 electrode catheters)	4	0
Faulty connexions	3	1
Mechanical and electrical faults	16	5
Increased threshold for pacing requiring unit removal	3	8
Total†	105	23

* See text.
† Includes 4 units implanted at other hospitals.

extended, since it has been found that the power required for artificial pacing was originally overestimated. A unit with a pulse duration of 1·2 msec. with a mean drain on the batteries between 20 and 60 μA is now in use, and the batteries are expected to last between 3 and 5 years. Less power is required for endocardial pacing (4 microjoules) in contrast to epicardial pacing (25 microjoules). The unit discharge rate occasionally increased shortly after implantation due to the higher temperature of the body, and this fault has now been eliminated. It is a remarkable fact that only one instance of pacemaker failure has been directly responsible for a patient's death. Fortunately, when a unit fails, the patient's idio-ventricular rhythm has taken over, with the exception of one patient. Stokes-Adams attacks requiring emergency treatment are likely to occur, however, even in those patients who have never had them before. Emergency replacement of a pacemaker is rare now that we change the units immediately the discharge rate falls by 10 to 15 impulses a minute.

Breakage of wires joining the pacemaker to the heart occurred occasionally when braided steel was used. Replacement by coiled stainless steel wire, forming a continuous loop from the pacemaker to the heart and back again, and avoiding any junctions or change of material that would predispose to corrosion, has met with complete success. No fault in a wire has developed since this method came into use 2 years ago.

The principal advantage of the endocardial-jugular system with the external pacemaker is the ease of insertion of the electrode catheter and servicing of the units. The power of the pacemaker is checked every 3 months in our out-patients' department and the unit is easily changed when necessary. The chief disadvantages are the relative ease with which the endocardial catheter can be pulled out of position and the local area of infection around the catheter in the neck and the occasional septicæmia. The buried axillary-jugular system avoids these complications.

Mortality of Long-term Pacing

Of the 88 patients treated by long-term pacing, 21 (23·8%) have died, and Table XII summarizes the causes of death. Eight patients died in the post-operative period following insertion of an abdominal-epicardial system of pacing. While some deaths may be attributed to the risks of an operation in a group of elderly patients, the commonest early cause of death was ventricular fibrillation: this was particularly liable to occur in patients who had returned to sinus rhythm, following the establishment of satisfactory pacing, and only occurred with the abdominal-epicardial system of pacing. The mortality of 23·8 per cent in the paced patients compares favourably with the mortality of 30 per cent in the less severely affected patients who were managed on medical treatment alone over the same period of time.

TABLE XII

Cause of Death in 21 Long-term Paced Patients

	No. of patients
Observed ventricular fibrillation	4
Cardiac infarction	2
Renal failure	1
Broncho-pneumonia and septicæmia	1
Pulmonary emboli	2
Fractured skull following fall	1
Congestive cardiac failure	2
Pacemaker failure	1
Cause of death unknown*	7

* Probably ventricular fibrillation.

Discussion

In our experience the method of choice for long-term pacing is the endocardial-jugular system with a buried pacemaker. This is contrary to the views held by Cole and Yarrow (1964) who considered on theoretical grounds that endocardial pacing on a long-term basis would be fraught with difficulties, particularly in relation to thrombus formation and dislodgement of the electrode within the heart. The complications following insertion have been remarkably few when they are compared with those of the abdominal-epicardial system, though the two groups are not entirely comparable;

for example, most of the abdominal units were covered with epoxy resin and those in the axilla with silicone rubber, and the endocardial group were treated when considerable experience had been accumulated, particularly regarding drug management.

We have now come to rely entirely upon the endocardial-jugular system with a buried pacemaker except in those patients in whom endocardial pacing is contraindicated owing to valve disease and a risk of bacterial endocarditis, or, rarely, when a satisfactory "wedged" position of the electrode tip cannot be obtained in the right ventricle. Thrombotic and septicæmic episodes do not appear to be a significant hazard of the endocardial-jugular system with a buried pacemaker implanted under satisfactory conditions. This is in contrast to the endocardial-jugular system with an *external* pacemaker, where septicæmia occurred in 4 patients, presumably from the area of infection at the point of exit of the catheter in the neck, especially when the catheter had to be repositioned in the presence of an obvious infected area around the exit site; this may perhaps be avoided if the endocardial catheter is exteriorized in the axilla, placing a greater distance between the jugular vein and the infected area. The rise of threshold that occurs with systems using epicardial electrodes has been less with the endocardial electrodes; this is related to the lower incidence of rejection and sepsis with the endocardial system and is similar to the experiences of Furman *et al.* (1961), and Landegren and Biörck (1963), who have also left electrode catheters in the right ventricle for many months. The endocardial-jugular system with the buried pacemaker is relatively easily implanted with only minor disturbances to the patient, so that age and physical condition are no longer a contraindication to operation. Improvement in a patient's state was almost invariable with any method of pacing, but has sometimes been overshadowed by the complications that have arisen during the maintenance of long-term pacing by the abdominal-epicardial systems.

The preponderance of patients with a right bundle-branch block pattern electrocardiogram, whether conducted or idio-ventricular, is striking (70%), and may be related to the relatively compact arrangement of the right bundle being more easily interrupted than the left by localized fibrotic lesions in the conduction fibres, which is often the only abnormality found at necropsy. In fact it was the demonstration of the absence of coronary disease by Professor Crawford (Leatham *et al.*, 1956) in our first patient that made us realize the importance of developing techniques for permanent pacing. The finding of non-specific fibrosis of the conducting tissue, of unknown ætiology, as the commonest cause of chronic atrio-ventricular block is in agreement with the views of Shirley Smith and Zoob (1961), Johansson (1961), and Dittmar *et al.* (1962).

It is possible that many patients with heart block due to coronary disease die during their first or second Stokes-Adams attack, and only a few survive multiple attacks and are considered for artificial pacing. This may explain the findings of Penton *et al.* (1956) and Rowe and White (1958) who demonstrated that in patients with complete heart block those with Stokes-Adams attacks survived statistically twice as long as those without. Johansson (1961) also noted that the prognosis was quite good if the patients survived the first few months, which might imply that the coronary vessels were reasonably healthy in the survivors.

The high incidence of sepsis and rejections of the pacemaker in the abdominal-epicardial system may be related to a low grade infection introduced at the time of operation or to a local reaction to the unit when implanted within the rectus sheath. In favour of a local reaction being responsible is the complete absence of a similar reaction when the pacemaker is implanted in the axilla.

It is now realized that the presence of rheumatic valve disease contraindicates endocardial electrode pacing with an external pacemaker, since 3 patients developed bacterial endocarditis shortly after insertion of the jugular electrode catheter. This is probably related to the area of infection at the point of exit of the catheter in the neck, and it may be perfectly safe in future to use an *implanted* pacemaker in combination with an electrode catheter in the presence of rheumatic or congenital heart disease.

The incidence of ventricular fibrillation after pacing has been established is higher in those patients who have returned to sinus rhythm. All of the "long-term" patients who died from ventricular fibrillation had resumed A-V conduction with the production of parasystole, but the pace-

makers in use at the time were overpowered. Wiggers and Wégria (1940), Brooks *et al.* (1955), and Lown, Amarasingham, and Neuman (1962) have shown that there is a period in late systole, extending for 30 msec. before the apex of the T wave in the cardiogram, during which ventricular fibrillation may be provoked by a suitable stimulus. When the pacemaker is not completely controlling the heart rate following a return to sinus rhythm, a proportion of the impulses will fall in the vulnerable periods. Since a much larger number of patients have continued to survive with parasystole, some additional factors must be required to reduce the threshold to triggering ventricular fibrillation (Chardack *et al.*, 1963; and Johansson *et al.*, 1963). Since ventricular fibrillation while pacing with endocardial systems has not been seen, except following cardiac infarction in 1 per cent not in this series, and all observed cases have occurred with the abdominal-epicardial systems within a few days of operating, it is possible that damage to the myocardium at operation may temporarily lower the threshold for the electrical impulse to produce ventricular fibrillation, especially in the presence of anoxia from post-operative chest complications. This is confirmed by the absence of ventricular fibrillation, precipitated during parasystole, as a late complication of epicardial pacing. Several instances of ventricular tachycardia have, however, been recorded when impulses fell on the T wave of ectopic beats some time after artificial pacing was started. Recently β-blocking agents have been used with success, to suppress ectopic beats when there appears to be a risk of ventricular arrhythmia. Any risk of pacemaker-induced ventricular fibrillation could be avoided by means of an atrial-controlled pacemaker (Nathan *et al.*, 1963) or by cessation of pacing during periods of sinus rhythm (Abrams, Hudson and Lightwood, 1960), with the addition of automatic response of the pacemaker to asystole (Johansson, 1961; Nicks, Stening, and Hulme, 1962), but these refinements may not prove necessary and will add to the frequency of unit failure.

Though all 5 patients who developed complete heart block following acute myocardial infarctions recovered normal A-V conduction after pacing, 4 died suddenly later. Since a recent report by Brown *et al.* (1963) showed that the commonest rhythm before the onset of ventricular fibrillation in patients with acute myocardial infarcts is a slow nodal rhythm, it may be worth while considering continuation of artificial pacing for several weeks after normal conduction has recovered, at a rate sufficient to suppress sinus rhythm. This would allow time for the conduction tissue to recover more fully.

For patients with chronic atrio-ventricular block with recurrent Stokes-Adams attacks or heart failure, who have failed to respond to medical treatment, artificial pacing offers possibilities of return to a more or less normal way of life: but the problems of maintenance and complications of long-term pacing have been numerous and this treatment cannot be embarked on lightly. However, 22 patients with endocardial-jugular systems and implanted pacemakers have done well with few complications.

Our current management of the complications of complete heart block is summarized in Table XIII.

TABLE XIII

LONG-TERM MANAGEMENT OF HEART BLOCK WITH SYMPTOMS

A: Inadequate cardiac output including cardiac failure and confusional states
(1) Digitalis (unless A-V block incomplete). Diuretics and sympathomimetic amines
(2) Temporary pacing with endocardial system (preferably not jugular vein) and if good response proceed to 3
(3) Endocardial-jugular pacing with implanted pacemaker or, if necessary, abdominal-epicardial system with implanted pacemaker

B: Stokes-Adams attacks
(1) Sympathomimetic amines especially long-acting isoprenaline (saventrine)
(2) Endocardial-jugular pacing with implanted pacemaker

SUMMARY

The results are given of 4 years' experience of long-term pacing in 88 patients, and of temporary

pacing in 12 patients, all with complete heart block which had failed to respond to medical treatment. During the same period, 40 patients with less severe symptoms have been managed on medical treatment alone. During an average pacing period of 12·7 months, 21 (24%) of the long-term paced patients have died and the majority of the 67 survivors are relatively well and leading an active life. Of the temporarily paced patients, 7 (58%) have died, 4 of whom had developed complete atrio-ventricular block following an acute myocardial infarction. Of the 40 patients managed on medical treatment alone, 12 (30%) have died.

The 3 systems of artificial pacing are described, abdominal-epicardial, endocardial-jugular with an external pacemaker, and the endocardial-jugular with a buried pacemaker. The complications arising from the abdominal-epicardial system for long-term pacing have been high in contrast to the endocardial-jugular system with a buried pacemaker which is now our method of choice for long-term pacing.

The incidence of a right bundle-branch block pattern in the electrocardiogram was high. Coronary artery disease was not a common cause of chronic heart block as judged by clinical and post-mortem findings.

Treatment of Stokes-Adams attacks with sympathomimetic amines, without electrocardiographic diagnosis of the underlying rhythm, may be extremely hazardous when ventricular tachyarrhythmias are responsible.

Addendum

Further experience, since May 1964, has confirmed our confidence in the endocardial-jugular system with a buried axillary pacemaker as the method of choice for long-term pacing. We now (February 1965) have 35 patients with this system of pacing who are leading a normal life.

Two electrode catheters have cracked 10 months after installation, resulting in a loss of power for pacing, and they had to be replaced. It is realized that this fault requires correction and we are now developing our own electrode catheter. The pacemakers of the earlier design have continued to become prematurely exhausted and 14 have required replacement, but the latest unit (Type X) is satisfactory.

The aftercare of a large number of artificially paced patients presents a considerable strain upon the technical and nursing staff. Many problems requiring emergency treatment can be avoided by careful supervision of patients in an out-patient "pacemaker" clinic. The organization of this clinic and the early detection of faults and their correction is to be published (Bluestone, R., Harris, A., Davies, G., 1965, *Brit. med. J.*, in the press). Our experience with implanted pacemakers, particularly the technical considerations, is also to be published (Davies G., and Siddons, H., 1965, *Thorax*, in the press).

The abdominal and axillary pacemakers are now obtainable from Devices Ltd., Welwyn Garden City, Herts., England.

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